

Guillain-Barré Syndrome and Postbariatric Surgery Polyneuropathies

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ABSTRACT

Background: Postbariatric surgery poly-neuropathies (BSP) are infrequent events. However, with the number of gastric bypasses performed each year increasing, the incidence of BSP is expected to increase as well. The long-term sequelae of BSP worsen with delays in diagnoses. Therefore, early evaluation, recognition, and treatment are important in minimizing morbidity and mortality.

Methods: We present the case report of a patient who developed a poly-neuropathy after a laparoscopic Roux-en-Y gastric bypass.

Results: The patient's symptoms were ultimately determined to result from Guillain-Barré syndrome. Symptoms resolved with proper treatment.

Conclusion: Characteristic differences exist between the various surgery poly-neuropathies. With proper evaluation, this determination will aid in prompt and appropriate treatment and will prevent morbidity and mortality.

Key Words: Bariatric surgery poly-neuropathy, Guillain-Barré syndrome, laparoscopic Roux-en-Y gastric bypass.

INTRODUCTION

Poly-neuropathies are an uncommon condition and may result from several causes. These include drugs, metabolic disorders, environmental exposure, vitamin deficiencies, conversion disorders, and inflammation. Drug-related neuropathies, ie, exposure to cisplatin, chloramphenicol, dapsone, isoniazid, metronidazole, nitrofurantoin, pyridoxine, amiodarone, tetanus toxoid, and diphtheria toxin, are the most common type.¹ Metabolic disorders that can cause poly-neuropathies include diabetes mellitus, uremia, hypothyroidism, hepatic failure, polycythemia, and porphyria.² Environmental exposure, such as to toxins (acrylamide, carbon disulfide, ethylene oxide, and carbon monoxide), metals (mercury, gold, and thallium), and alcohol can also result in a poly-neuropathy.³

Acquired deficiencies in thiamine (vitamin B₁) and cyanocobalamin (vitamin B₁₂) are the most common causes of postbariatric surgery poly-neuropathies (BSP).^{4–8} Inflammatory causes of BSP include Guillain-Barré syndrome (GBS), myasthenia gravis, human immunodeficiency virus associated poly-neuropathy, lupus erythematosus associated poly-neuropathy, and chronic active hepatitis associated poly-neuropathy.⁹

The literature has revealed that the incidence of BSP is 0.06% (5.9 cases per 10 000 operations).⁴ Similarities between thiamine deficiency and GBS include the presence of protracted vomiting, weakness, and hyporeflexia. The long-term sequela of each disease varies, but worsens with delays in diagnoses and treatment. Therefore, establishing the correct diagnoses early will minimize morbidity and mortality.

CASE REPORT

A 40-year-old female with a body mass index of 60, who met the National Institutes of Health criteria for weight loss surgery, underwent an uneventful antecolic antegastric laparoscopic Roux-en-Y gastric bypass. Postoperatively, she experienced an episode of nausea and vomiting that resolved spontaneously. She was discharged home on postoperative day 2.

Subsequently, the patient was readmitted 3 times in 5

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months for protracted nausea, vomiting, lethargy, and weakness. On her first admission, her physical examination and electrolytes were normal. An upper gastrointestinal study with small-bowel follow-through and an esophagogastroduodenoscopy were negative for small-bowel obstruction, anastomotic stricture, and marginal ulcer. At her second admission, on postoperative week 8, she was managed conservatively with intravenous fluids and electrolyte replacement. She was readmitted 4 weeks later with an exacerbation of her nausea, vomiting, and right upper quadrant tenderness. A CT scan of the abdomen obtained during this admission revealed cholelithiasis. Although her preoperative ultrasound had been negative, symptomatic cholelithiasis was considered as a possible cause of her symptoms. She, therefore, underwent an uneventful laparoscopic cholecystectomy. After her surgery, she was noted to have inadequate oral intake and persistent nausea and vomiting; therefore, a PICC line was placed for home TPN with vitamin and nutritional supplements. Prior to discharge, the patient noted the onset of mild numbness and tingling extending from her lower extremities to her abdomen. She was discharged home on postoperative day 2.

At home, her symptoms rapidly progressed within 1 week from bilateral lower extremity cramping and burning to an inability to walk or stand. The patient underwent a neurologic workup. A head CT, MRI, cerebral spinal fluid smear and culture were negative. She was referred to a neurologist at a rehabilitation facility for further evaluation.

The neurologist's evaluation showed bilateral 3/5 motor strength in her distal lower extremities, markedly decreased sensation from the middle of her thigh distally, decreased vibration sense in her lower extremities, and absent ankle and knee-jerk reflexes. Laboratory studies included a normal CBC and electrolytes, serum alkaline phosphatase of 164 (normal, 25 to 150), serum AST of 274 (normal, 0 to 40), serum ALT of 145 (normal, 0 to 40), glycosylated hemoglobin of 4.3 (normal, 4.5 to 5.7), negative Lyme titer, Vitamin B12 level of 691 (normal, 211 to 911), folic acid level of 9.7 (normal >5.4) and serum albumin of 3.5 g/dL.

Nerve conduction studies (NCV) revealed florid fibrillations and positive waves in the gastrocnemius, peroneous longus, and adductor longus muscles indicating a significant sensorimotor distal polyneuropathy. These findings were consistent with the axonal form of GBS. The patient was prescribed Gabapentin 100 mg at night, and continued her rehabilitation with physical therapy. Her symp-

toms gradually improved; however, she required the assistance of a walker for 6 months before her symptoms completely resolved.

DISCUSSION

GBS is an acute postinfectious autoimmune syndrome affecting the peripheral nerves and resulting in ascending symmetrical motor and sensory deficits. Classic symptoms on presentation, after bariatric surgery, include protracted nausea, vomiting, and lower extremity weakness.^{4,5} Generalized weakness can progress over days to weeks. GBS can also present with cranial nerve deficits and respiratory compromise. The patient may describe a viral or bacterial infection 1 week to 3 weeks prior to symptoms.

Infection with *Campylobacter jejuni* specifically has been linked to the initiation of symptoms.¹⁰ Other associated infections include Epstein-Barr virus, varicella-zoster virus, human immunodeficiency syndrome, and *Mycoplasma pneumoniae*.¹¹⁻¹³ The diagnosis is made based on history, physical examination, electromyography, nerve conduction studies, and cerebral spinal fluid test results.

Our patient underwent an extensive evaluation by a neurologist. The findings were consistent with GBS. Paulson et al¹⁴ found that 4 of 6 patients in his study, in addition to nutritional deficiencies, were found to have GBS by electromyography (EMG). Symptoms included confusion, inappropriate behavior, profound weakness, and paraplegia. Feit et al¹⁵ reported on 2 patients with postgastric partitioning poly-neuropathy. Both patients had symptoms of hysteria, protracted nausea and vomiting, and severe paraplegia. Diagnostic workups, including a negative cerebral spinal fluid aspirate, were inconclusive for both patients. However, both patients had an EMG that documented a demyelinating poly-neuropathy. Chang et al⁴ reported on 2 patients who underwent open gastric bypass and were postoperatively diagnosed with GBS. One patient had an inconclusive finding on EMG, but was assigned a diagnosis of GBS. Treatment was initiated with plasmapheresis followed by intravenous immunoglobulin (IVIG). Once started on antipsychotics (for her dysesthesias), this patient developed psychosis. The medication was discontinued and the psychosis resolved.

The second patient had typical GBS findings on EMG and NCV studies. She was treated with IVIG, which resulted in marked improvement. Sarris et al¹⁶ identified 3 of 4 patients, and Abarbanel et al¹⁷ identified 4 of 20 patients who had post-BSP treated with vitamin supplementation. Over 50% of their patients had an unknown cause of their

poly-neuropathy and developed residual weakness despite vitamin supplementation. Teitleman et al¹⁸ report on a patient treated with thiamine with resolution of symptoms. Machado et al¹⁹ report similar findings in a postbariatric surgery patient with resolution after treatment with immunoglobulin and vitamin supplementation.

GBS has many variants distinguished by physical symptoms and syndrome progression. Several diagnostic studies are used to evaluate GBS. These studies, guided by the patient's history and presentation, include a cerebral spinal fluid (CSF) aspiration to assess elevated protein (>0.55 g/L), a thyroid panel, rheumatology profiles, vitamin B1, vitamin B12, folic acid, glycosylated hemoglobin, erythrocyte sedimentation rate, rapid plasmin reagin, immunoelectrophoresis of serum protein, a heavy metal test, and serum auto antibodies against glycolipids on the surface of *C. jejuni*.¹⁰ Electromyography in GBS shows abnormalities consistent with demyelination.

Treatment options include physical therapy, occupational therapy, and speech therapy. ICU monitoring for respiratory failure and deep venous thrombosis (DVT) prophylaxis for prolonged immobility are also indicated. Specialty consults may include neurology, critical care, pulmonology, and surgery. Medical therapy may include IVIG, plasma exchange, nonsteroidal anti-inflammatory drugs, opioids, and Gabapentin.¹⁷ Van der Meche et al²⁰ showed that IVIG is indicated in patients with sensory variant GBS with prolonged, unresolved symptoms, but plasmapheresis has been shown to be equally effective in treating GBS. Immobility will require deep venous thrombosis prophylaxis, rehabilitation, and frequent turning to prevent decubitus ulcers. As an outpatient, the patient should be followed as needed by neurology, physical therapy, occupational therapy, and speech therapy.

Seventy-five percent to 85% of patients will have complete recovery.^{21,22} Fifteen percent to 20% will have residual motor and sensory deficits, and 1% to 10% will be severely disabled.^{21,22} Two percent to 12% of patients will die from complications (eg, ventilator-associated pulmonary infection) associated with GBS.²² Recovery is dependent on many factors including timeliness of diagnosis, acuity of the onset of symptoms, age of patient, concurrent infections, and need for ventilator support.^{21,22}

The differential diagnosis of BSP can be challenging. GBS and its variants²³ can be confused with thiamine deficiency with manifestations of dry beriberi and Wernicke's encephalopathy. Both present with lower extremity weakness, ataxia, and ophthalmoplegia. Differentiating factors include history and the results of EMG, NCV, and CSF.

CONCLUSION

BSP is a complication that may increase in incidence as the number of bariatric surgery procedures increase. Understanding its clinical presentation, differential diagnoses, proper evaluations, and treatment are necessary to minimize morbidity and mortality. It is prudent to evaluate for nutritional deficiencies (vitamin levels, prealbumin, transferrin, retinol binding protein), provide vitamin B1 and B12 supplementation, and treat according to findings obtained during a comprehensive neurological and psychological examination. A high index of suspicion and early recognition are essential in preventing morbidity and mortality.

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